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Assessment of Cochlear Trauma During Cochlear Implantation Using Electrocochleography and Cone Beam Computed Tomography

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Objective: To assess cochlear trauma during cochlear implantation by electrocochleography (ECoG) and cone beam computed tomography (CBCT) and to correlate intraoperative cochlear trauma with postoperative loss of residual hearing.
Methods: ECoG recordings to tone bursts at 250, 500, 750, and 1000 Hz and click stimuli were recorded before and after insertion of the cochlear implant electrode array, using an extracochlear recording electrode. CBCTs were conducted within 6 weeks after surgery. Changes of intraoperative ECoG recordings and CBCT findings were correlated with postoperative threshold shifts in pure-tone audiograms.
Results: Fourteen subjects were included. In three subjects a decrease of low-frequency ECoG responses at 250, 500, 750, and 1000 Hz occurred after insertion of the electrode array. This was associated with no or minimal residual hearing 4 weeks after surgery. ECoG responses to click stimuli were present in six subjects and showed a decrease after insertion of the electrode array in three. This was associated with a mean hearing loss of 21 dB in postoperative pure-tone audiograms. Scalar dislocation of the electrode array was assumed in one subject because of CBCT findings and correlated with a decrease of low-frequency ECoG responses and a complete loss of residual hearing.
Conclusion: Hearing loss of $\leq$11 dB is not associated with detectable decrease in ECoG recordings during cochlear implantation. However, in a majority of patients with threshold shifts of $>$11 dB or complete hearing loss, an intraoperative decrease of high- or low-frequency ECoG signals occurs, suggesting acute cochlear trauma. **Key Words:** Cochlear implant—Cochlear implantation—Electrocochleography—Hearing preservation—Residual hearing.

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Minimizing trauma to cochlear structures should be attempted in all cochlear implant recipients nowadays (1). This allows preservation of residual hearing, leads to better cochlear implant performance in standard cochlear implant recipients without residual hearing (2–4), and enables access to possible future treatment options that are dependent on remaining intact cochlear structures. However, despite the degree of progress regarding atraumaticity, cochlear implantation still leads to postoperative hearing loss in a majority of patients (5–7). The underlying reasons are controversial. Besides cochlear trauma during surgery, postoperative mechanisms seem to cause deterioration of cochlear function (8–11). Therefore, methods to evaluate and monitor cochlear trauma during cochlear implantation could help to better understand the correlation between cochlear trauma and loss of cochlear function.

Usually, cochlear trauma is assessed by the grade of preserved residual hearing after cochlear implantation (1). However, a distinction between hearing loss caused by surgical trauma and hearing loss because of postoperative mechanisms is not possible using this approach. Imaging techniques have been proposed to visualize the electrode array and thereby give indirect information about trauma to cochlear structures. Besides high-resolution computed tomography (3,12), cone beam computed tomography (CBCT) (13) has gained increasing support as it is a quick procedure and the radiation dose seems to be lower than with high-resolution computed tomography (14). Furthermore, previous reports have shown high reliability for correctly detecting scalar position of the electrode array within the basal turn of the cochlea (15–18).

Still, the optimal tool to assess cochlear trauma during the cochlear implantation itself would allow monitoring of

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cochlear function during surgery. Electrocochleography (ECoG) has shown great potential in this regard and has been conducted in animal studies (19–24) as well as during and after cochlear implantation in humans (9–11,25–29). To our knowledge, four reports have correlated intraoperative ECoG findings with postoperative hearing preservation rates (9–11,28). One report showed a close relationship between trauma detected by intraoperative ECoG and postoperative hearing preservation rates (28); whereas the other three suggested limited predictive value of low-frequency ECoG recordings regarding postoperative hearing loss.

This study was conducted as a follow-up to our first study (10), in which we evaluated changes of low-frequency ECoG signals at suprathreshold intensities during cochlear implantation. The primary aim was to evaluate if the predictability of postoperative hearing changes by intraoperative ECoG recordings could be increased by the addition of ECoG recordings to high-frequency acoustic stimuli and/or threshold determinations. The secondary aim was to correlate scalar dislocation according to CBCT findings with intraoperative ECoG findings.

METHODS

Included subjects were adult patients (>18 yr) undergoing cochlear implantation at the University Hospital of Zurich. The study was performed with the approval of the Ethical Committee of Zurich (KEK-ZH 2013-0317) and in concordance with the Helsinki Declaration. All subjects provided written informed consent before surgery. As cochlear implant devices the Cochlear Nucleus CI-512, CI-422 and CI-522 were used.

Pure-tone audiograms were conducted according to ISO 8235-1 within 3 months before surgery and 4 weeks postoperatively. The pure-tone average (PTA) was calculated from the threshold values at 250, 500, 1000, 2000, and 4000 Hz. Hearing loss related to the cochlear implantation was defined as the difference between pre- and postsurgical PTAs. The maximum output of the audiometer plus 5 dB was entered if no responses were present at the maximum output.

Surgery and Recording Setup

Before surgery, an insert earphone (Biologic Systems, Mundelein, IL, U.S.A.) and a probe microphone (ER-7C, Etymotic, Inc., Elk Grove Village, IL, U.S.A.) were placed in the ear canal. Electrodes (Neurosign, Magstim Co., Wales, U.K.) were placed in the contralateral preauricular region (''negative'') and on the forehead (''ground''). Next, an anterior mastoidectomy and posterior tympanotomy via the facial recess were performed and the round window completely visualized. A standard needle electrode (20 × 0.3 mm, Neurosign, Magstim Co.) was then used as the recording electrode and was placed anterior-superior of the round window. Once impedances were <10 kOhm on all electrodes, baseline ECoG recordings were performed. With the recording electrode left in an unchanged position, an anterior-inferior cochleostomy or a round window insertion following soft surgical principals was conducted. After full insertion of the cochlear implant array and sealing of the insertion site with periosteum, the ECoG recordings were repeated. The recording electrode was then removed and the surgical incision closed in layers.

Acoustic Stimulation/Recording and ECoG Recording

The Navigator Pro stimulation/recording device (Biologic Systems) was used for acoustic stimulation and recording. Responses to 400 low-frequency tone bursts or 400 clicks—representing the high-frequency acoustic stimulus—with alternating starting phases were filtered (the high pass filter was set at 10 Hz, the low pass filter at 5000 Hz) and then averaged after rejection of artifacts. A value of 47.5 uV was selected for artifact rejection. The tone burst rise and fall times were 2 cycles shaped by a Blackman window. The plateau phase was 4 cycles at 250 Hz, 10 cycles at 500 Hz, 14 cycles at 750 Hz, and 20 cycles at 1000 Hz. The recording window for tone bursts was 32 ms, starting 4 ms before stimulus presentation. For click stimuli, the recording window was 10.66 ms, starting 1 ms before stimulus presentation. The sampling rate was 8000 Hz for 250, 500, 750 Hz, and click stimuli and 16,000 Hz for 1000 Hz stimuli.

Maximum sound pressure was 85 dB nHL at 250 Hz, 95 dB nHL at 500 Hz, 100 dB nHL at 750 Hz and 1000 Hz, and 95 dB nHL for click stimuli. At the frequency with the most robust ECoG response to tone bursts at the maximum intensity, a level series in 5 dB descending steps was conducted until the visually detected threshold. This protocol was performed before and after insertion of the cochlear implant electrode array.

For recording of the acoustic signal, the probe microphone placed in the ear canal was connected to a dynamic signal analyzer (CoCo-80, Crystal Instruments Corporation, Santa Clara, CA, U.S.A.). Further postprocessing of the acoustic signal was done with the Engineering Data Management software (EDM, Version 2.2.0.3; Crystal Instruments Corporation).

Data Analysis

To export data from the AEP software (Biologic Systems, Mundelein, IL, U.S.A.), the AEP to ASCII software (Biologic Systems) was used. Data postprocessing was done using MATLAB (MathWorks, Inc., Natick, MA, U.S.A.) and GraphPad Prism V5.04 (GraphPad Software, Inc., San Diego, CA, U.S.A.).

The average response from condensation and rarefaction phases was stored separately. To obtain the difference curve, the average response of both phases was subtracted; to obtain the alternating curve, the average response of both phases was added. The spectrum from each difference and alternating curve was determined from the FFT. To assess the spectrum of the ongoing ECoG response—defined as the part of the ECoG response that lasts for the duration of the acoustic stimulus after the compound action potential (CAP)—a time window from 9 to 23 ms was used. The amplitude of the ongoing ECoG response was determined as follows: the response amplitude at the stimulus frequency (i.e., fundamental frequency or first harmonic) in the difference curve and the response amplitude at the frequency of the second harmonic in the alternating curve were added. The sum was defined as the amplitude of the ongoing ECoG response. This definition has been used in multiple studies (27,30–32). An ongoing ECoG response was considered valid if the amplitude exceeded the mean noise floor plus 3 standard deviations (9,10,25,29–32). This means that less than 1% false-positive results can be expected. The mean noise floor was calculated from all bins within 100 Hz on each side of the assessed frequency starting 50 Hz away from the peak. If postprocessing revealed a lower threshold than was visually assumed during surgery, meaning that true threshold was not reached in the level series, then the lowest intensity measured minus 5 dB was considered threshold.

The CAP was assessed in the alternating curve. The presence of a CAP was determined visually by two experienced otorhinolaryngologists (A.H., D.V.), blinded to the pre- and postoperative residual hearing and CBCT findings of subjects.

Radiological Examination

Postsurgical imaging using CBCT was performed within 6 weeks after surgery using the 3D Accuitomo 170 (J. Morita Mfg. Corp., Kyoto, Japan). Imaging was performed with a tube current of 5 mA and a tube voltage of 90 kV. One 360-degree scan took 30.8 seconds. A field of view of 40 × 40 mm was selected. Images were reconstructed in 80-μm isometric voxels using the i.Dixel One Volume Viewer software (Version 2.6.0, J. Morita Mfg. Corp.).

The scalar localization of the cochlear implant electrode array was assessed by two experienced otorhinolaryngologists (A.H., D.V.), blinded to the intraoperative ECoG findings and hearing outcome of subjects.

RESULTS

Subject demographics are summarized in Table 1. Mean age was 51 years (range from 28 to 77 yr) and duration of deafness was >10 years in all subjects. Surgeries were performed between September 2014 and June 2015. No complications occurred during surgery. In the ear canal, mean difference in sound pressure between pre- and postinsertional recordings was 0.4 dB with a standard deviation of 1.6 dB.

Audiometric, radiological, and electrophysiological findings are summarized in Table 2. On the operated side, mean presurgical PTA of all subjects was 94 dB HL (range, 64–113 dB HL). Mean decrease in PTA 4 weeks after surgery was 15 dB (range, 4–32 dB). Eight subjects had a loss of ≤11 dB. Two subjects had a complete loss of residual hearing (Subjects 1 and 7). On the contralateral side, no subject showed a hearing loss of ≥10 dB during the study period.

Before insertion of the cochlear implant electrode array, all subjects except Subject 12 at 750 Hz showed relevant ongoing ECoG responses to tone bursts at 250, 500, 750, and 1000 Hz. The CAP responses to click stimuli were detectable in six subjects (1, 2, 4, 5, 8, and 11). Ongoing ECoG signals recorded under unchanged conditions showed a mean difference of 0.1 dB with a standard deviation of 1.2 dB. Therefore, changes of >2.5 dB (approximately 1.96 SD) between pre- and postinsertional ECoG recordings were considered relevant.

Figure 1 displays representative examples for pre- and postinsertional ECoG recordings. In Subject 6 (Fig. 1A), the ongoing ECoG signals remained almost unchanged after full insertion of the electrode array, whereas in Subject 10 (Fig. 1B) a decrease was detectable. Such a pattern of decrease of the ongoing ECoG signals at all recorded frequencies after insertion of the electrode array occurred in Subjects 7, 10, and 13.

Postinsertional CAP responses to click stimuli showed a decrease in three out of six subjects when compared with preinsertional CAP responses. In Subject 1 (Fig. 1C), the CAP response to click stimuli was completely lost, in Subjects 2 and 11 (Fig. 1D), there was a decrease in amplitude.

Overall, on the basis of these findings, we assume that gross cochlear trauma occurred in Subjects 7, 10, and 13, whereas in Subjects 1, 2, and 11, the cochlear trauma seemed to be limited to the high-frequency regions. The correlation between hearing outcome 4 weeks after surgery and intraoperative cochlear trauma is summarized in Figure 2.

In the level series, four subjects showed an increase of the detection threshold of 10 dB (Subjects 7, 9, 10, and 12). This was associated with a complete loss of residual hearing in Subjects 7 and 10 and a hearing loss of 5 and 4 dB in Subjects 9 and 12, respectively.

Figure 3 displays the level series of the ongoing ECoG response at 750 Hz for subjects with and without decrease of low-frequency ECoG responses after insertion of the electrode array. Both groups showed a parallel slope of pre- and postinsertional responses as a function of level until threshold. The overall linear best fit slopes were not significantly different ($F = 2.19$, $p = 0.12$). The calculated predictive slope for all data equaled 0.96.

TABLE 1. *Subject demographics*

Subject No.	Age (yr)	Etiology of Hearing Loss	Side	Round Window Insertion	Cochlear Implant
1	58	Idiopathic	Right	Yes	Nucleus CI-422
2	34	Large vestibular aqueduct	Left	Yes	Nucleus CI-422
3	77	Idiopathic	Left	No	Nucleus CI-512
4	69	Idiopathic	Right	Yes	Nucleus CI-422
5	47	Idiopathic	Right	Yes	Nucleus CI-422
6	39	Fetal rubella infection	Left	No	Nucleus CI-512
7	53	Otosclerosis	Right	No	Nucleus CI-512
8	28	Idiopathic	Left	No	Nucleus CI-512
9	51	Idiopathic	Right	No	Nucleus CI-512
10	75	Otosclerosis	Left	No	Nucleus CI-512
11	55	Idiopathic	Right	Yes	Nucleus CI-522
12	37	Idiopathic	Right	Yes	Nucleus CI-522
13	41	Idiopathic	Left	Yes	Nucleus CI-522
14	52	Idiopathic	Right	No	Nucleus CI-512

TABLE 2. *Audiometric, radiological, and electrophysiological findings*

Subject No.	Preoperative PTA (dB HL)	Postoperative PTA (dB HL)	Hearing Loss (dB)	Scalar Dislocation (CBCT Finding)	Change in Mean Low-frequency ECoG Response (dB)	Change in High-frequency ECoG Response (dB)	Frequency of Level Series (Hz)	Change in Detection Threshold of ECoG Response (dB)
1	89	121	32	No	0.1	Loss of response	750	0
2	93	100	7	No	1.3	Decrease	750	0
3	101	112	11	No	0.3	No response	1000	0
4	76	104	28	No	4.1	No decrease	750	5
5	78	89	11	No	11.1	No decrease	1000	−15
6	96	106	10	No	0.9	No response	250	−20
7	97	121	24	Yes	−4.3	No response	750	10
8	102	112	10	No	5.6	No decrease	750	0
9	110	115	5	No	1.6	No response	750	10
10	103	118	15	No	−4.4	No response	750	10
11	64	88	24	No	4	Decrease	750	0
12	99	103	4	No	6.1	No response	1000	10
13	113	119	6	No	−4.3	No response	250	0
14	99	117	18	No	5.5	No response	750	-5

CBCT indicates cone beam computed tomography; ECoG, electrocochleography; PTA, pure-tone average at 250, 500, 750, 1000, 2000, and 4000 Hz.

According to CBCT findings, scalar dislocation of the electrode array from the scala tympani into the scala vestibuli occurred in Subject 7. This was associated with complete loss of residual hearing and a decrease of low-frequency ECoG responses. In all other subjects, the position of the electrode array was judged as being inside the scala tympani in the basal turn. Both otorhinolaryngologists agreed on the scalar location of the electrode array in all patients. Figure 4 displays the CBCT findings of Subjects 6 and 7 as examples.

DISCUSSION

The mechanisms responsible for loss of residual hearing after cochlear implantation are controversial. Besides acute cochlear trauma during surgery, postoperative mechanisms seem to influence hearing preservation. Early inflammatory responses (33) or endolymphatic hydrops (34) could lead to hearing loss during the early postoperative phase, whereas tissue responses (8,35) and excitotoxicity because of electrical stimulation (36) could, among other factors, lead to delayed loss of residual hearing within months. Therefore, to further explore the correlation between trauma during surgery and postoperative hearing loss and gain more insight regarding the incidence of postoperative mechanisms, a tool to assess cochlear trauma during surgery is needed.

Animal studies have demonstrated that a decrease of the ongoing ECoG at low frequencies is a reliable marker for intracochlear trauma during insertion of an electrode (19–24,37). In cochlear implant recipients, findings thus far have suggested that a decrease of low-frequency ECoG signals at suprathreshold intensities during surgery results in hearing loss of >10 dB or complete hearing loss in a majority of patients (9,10). However, the data showed additionally that changes in low-frequency ECoG recordings during cochlear implantation do not directly translate into postoperative results for hearing threshold levels, and unchanged low-frequency ECoG responses during surgery do not exclude postoperative hearing loss (9–11,25). Two mechanisms could be responsible: 1) cochlear trauma that is not detectable by low-frequency ECoG recordings at suprathreshold intensities leads to postoperative hearing loss or 2) postoperative mechanisms not associated with cochlear trauma during surgery are responsible for loss of residual hearing in a considerable amount of cochlear implant recipients.

Therefore, to investigate whether in cochlear implant recipients ECoG recordings at higher frequencies or at lower intensities increase the predictability regarding postoperative hearing loss, we added ECoG recordings to acoustic click stimuli, which stimulate high-frequency regions of the cochlea between 1000 and 4000 Hz, and conducted a level series until the detection threshold of the ongoing ECoG response was reached.

The low-frequency ECoG recordings at suprathreshold intensities confirmed previously published results. Three subjects (Subjects 7, 10, and 13) showed a decrease of the

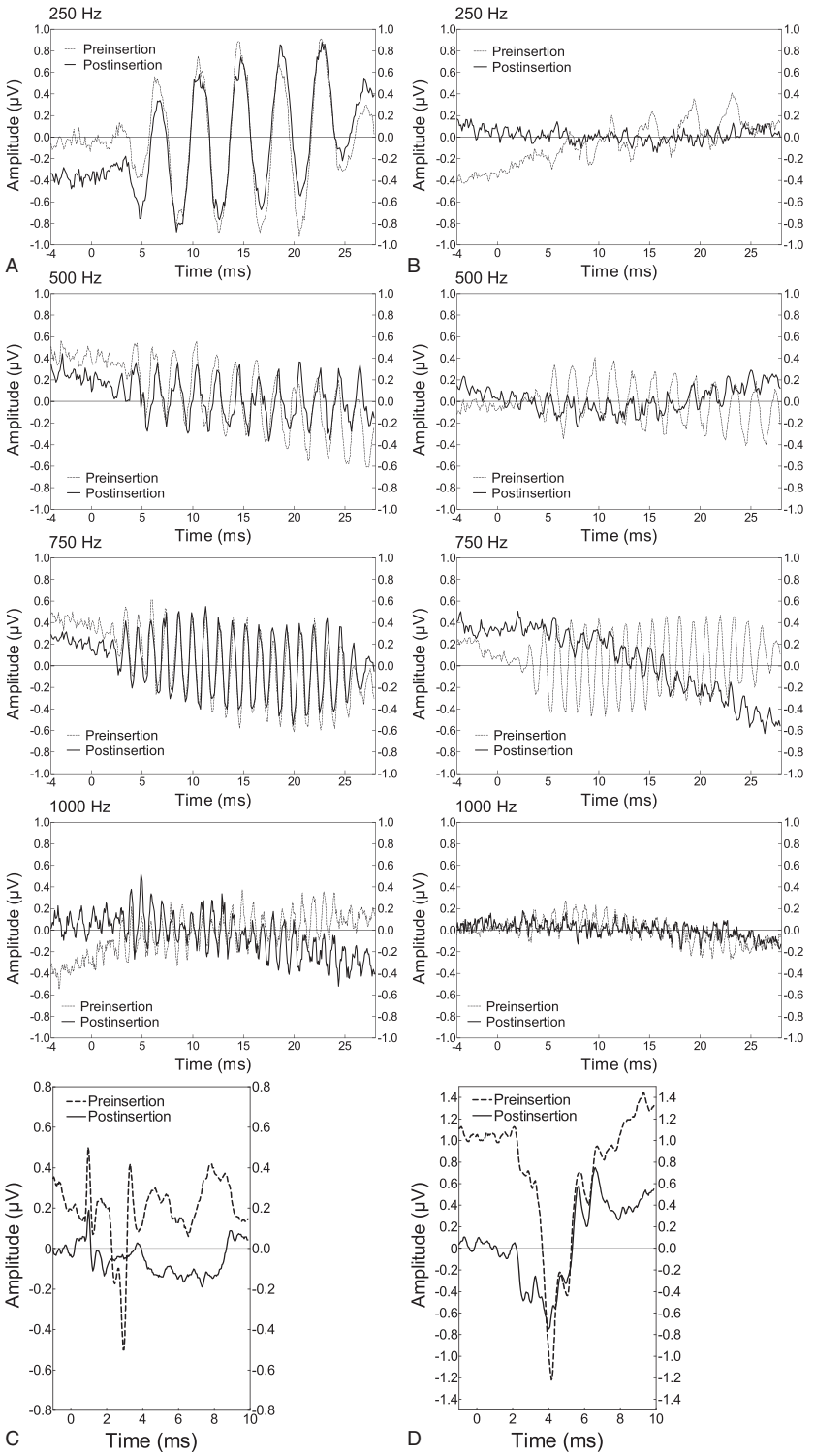

FIG. 1. Examples for pre- and postinsertional ECoG recordings to low-frequency tone bursts and click stimuli. *A*, *B*, The difference curves before and after insertion of the cochlear implant electrode array in Subject 6 (*A*) and Subject 10 (*B*) at 250, 500, 750, and 1000 Hz at maximum intensity are shown. In Subject 6 the responses remained almost unchanged whereas in Subject 10 a decrease of ECoG signals is visible. Such a decrease probably represents gross cochlear trauma. *C*, *D*, Two examples for patients in whom trauma to high-frequency regions of the cochlea during insertion of the cochlear implant electrode array was assumed. In both subjects, low-frequency ECoG recordings showed no decrease. The alternating curves in response to acoustic click stimuli are shown. In Subject 1 (*C*), a clear CAP was visible in preinsertional recordings. In postinsertional recordings, it was completely lost. This finding was associated with hearing loss of 32 dB, resulting in a complete loss of residual hearing. In Subject 11 (*D*), the CAP was markedly reduced in postinsertional recordings. This finding was associated with a postoperative hearing loss of 24 dB. CAP indicates compound action potential; ECoG, electrocochleography.

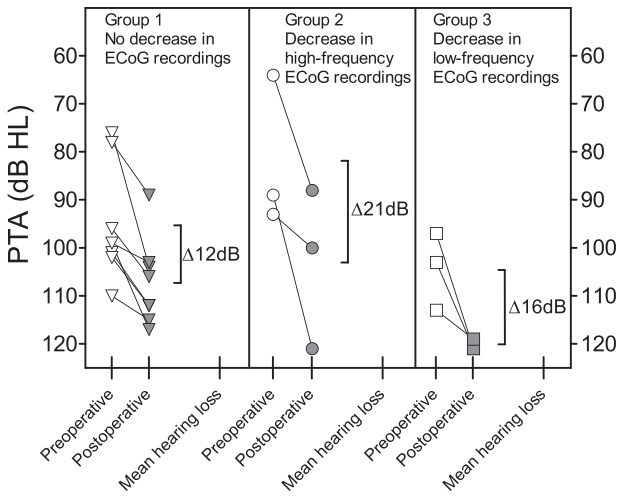

FIG. 2. Hearing outcome 4 weeks after surgery in correlation to intraoperative ECoG findings. The pre- and postoperative PTAs and the resulting hearing loss for subjects without detectable trauma in ECoG recordings (Group 1), with a decrease in high-frequency ECoG recordings (Group 2), and with a decrease in low-frequency ECoG recordings (Group 3) are shown. Group 1 showed a mean hearing loss of 12 dB with six out of eight subjects having a hearing loss $\leq$11 dB. Pure-tone audiograms in Group 2 revealed a mean hearing loss of 21 dB, including one subject with complete hearing loss (Subject 1). In Group 3 the mean hearing loss was 16 dB, resulting in a mean postoperative PTA of 120 dB HL (maximum 121 dB HL). PTA indicates pure-tone average.

ongoing ECoG response at all recorded frequencies at the maximum intensity level. All three subjects showed no (Subject 7) or minimal (Subjects 10 and 13) hearing preservation. This further confirms our assumption that in patients with a decrease of the ongoing ECoG response at low frequencies, preservation of residual hearing seems unlikely.

If present, the mean decrease of the ongoing ECoG signal amplitude in the low frequencies was an average of 4.3 dB for all three patients. This suggests that even in the patient of gross cochlear trauma, low-frequency ECoG responses are not immediately lost. This was also true for Subject 7, who not only had a decrease of the low-frequency ECoG response but also a scalar dislocation based on the CBCT findings. Furthermore, these findings show that a decrease of low-frequency ECoG responses probably represents relevant trauma but is not associated with a scalar dislocation in all patients. However, this case series is too small to fully elucidate the correlation between radiological and electrophysiological findings.

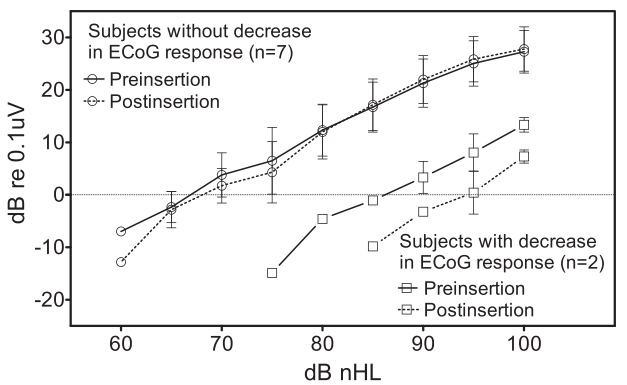

FIG. 3. Level series of ECoG recordings at 750 Hz are shown for subjects with and without decrease in postinsertional low-frequency ECoG recordings. In both groups, a parallel trend of the pre- and postinsertional level series can be observed. This suggests that changes of ECoG signals at high intensities resemble changes at lower intensities.

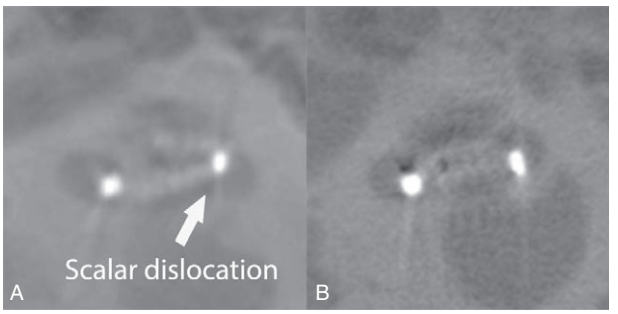

FIG. 4. CBCT findings of Subjects 6 and 7. *A*, The findings in Subject 7 suggest a dislocation of the cochlear implant electrode array from the scala tympani into the scala vestibuli. *B*, CBCT findings of Subject 6 are shown as a representative example for a correct position of the electrode array. CBCT indicates cone beam computed tomography.

High-frequency ECoG responses to click stimuli seem to add information about cochlear trauma to low-frequency ECoG recordings alone. High-frequency ECoG responses were detectable in six subjects, and of those three (Subjects 1, 2, and 11) showed a loss or decrease of the CAP after insertion of the cochlear implant electrode array during surgery. If present, the rate of detectable decrease was therefore higher in the high-frequency ECoG recordings than in the low-frequency ECoG recordings. All three subjects with detectable decrease in the high frequencies showed no decrease of the ongoing ECoG response in the low frequencies. The pure-tone audiograms 4 weeks after surgery showed a marked hearing loss in 2 (Subjects 1 and 11) out of these 3 subjects. Additionally, acute cochlear trauma limited to the high-frequency cochlear regions seems to influence not only hearing preservation in high-frequency but also in low-frequency regions: the mean hearing loss at 250, 500, and 1000 Hz in Subjects 1 and 11 was 33 and 22 dB, respectively. Early inflammatory responses triggered by minor trauma during surgery could be a possible explanation (33).

Eight subjects showed no decrease in low- or in high-frequency ECoG recordings immediately after surgery. Of these eight subjects, none had a complete loss of residual hearing and six subjects showed a hearing loss of $\leq$11 dB. This fits well with previously published results, which suggest that threshold shifts of approximately 12 dB occur without surgical trauma because of mechanical changes alone caused by the placement of an electrode array into the cochlea (38–40).

However, as in previous studies (9–11,25) marked hearing loss occurred in patients without detectable decrease in intraoperative ECoG recordings (hearing loss of 28 dB in Subject 4 and of 18 dB in Subject 14). This means that postoperative mechanisms independent of surgical trauma play a role or that acute changes in cochlear function during surgery were not detectable by ECoG recordings. As reported in previous studies (9,10), some ECoG responses showed an increase after insertion of the cochlear implant electrode. In this series, such a finding was present in five subjects (Subjects 4, 5, 8, 12, and 14). Different reasons could be responsible. First, such an increase could represent a recording bias because of contact of the recording electrode near the round window with perilymph. In such a patient, postinsertional ECoG recordings would represent intra- rather than extracochlear ECoG recordings, which would explain an increase of the ECoG response in comparison to preinsertional ECoG recordings (25,27). Second, pressure changes within the scala tympani could be responsible for an increase of ECoG signals as a close relationship between the amplitude of the CM and the pressure difference between scala tympani and scala vestibuli has been demonstrated (41). Third, the ongoing ECoG response represents a combination of hair cell and neural responses. The amplitude of the ongoing ECoG response is dependent on whether hair cell and neural components are in phase or not. This means that depending on the relative phase, the different components can add or subtract from each other. Therefore, a decrease of one component can lead to either an increase or decrease of the ongoing ECoG response (9,37).

A factor influencing the sensitivity of ECoG recordings to detect cochlear trauma during surgery could be the residual cochlear function before surgery. In concordance with previously published results (10), all three subjects with a detectable decrease of the low-frequency ECoG responses had limited residual hearing before surgery (mean presurgical PTA 104 dB HL). By contrast, changes only detectable in high-frequency ECoG recordings were present in subjects with considerable residual hearing before surgery (mean presurgical PTA 82 dB HL). Therefore, the detectability of cochlear trauma in high- and low-frequency regions during surgery could be influenced not only by the extent of cochlear trauma but also by the residual cochlear function before surgery.

Threshold determinations of the ECoG response seem to be less sensitive regarding cochlear trauma than do changes at higher intensities only. The reason could be that changes of the ongoing ECoG signal at higher intensities resemble changes at intensities near threshold and therefore also threshold changes themselves. As even gross cochlear trauma (i.e., scalar dislocation in Subject 7) seems to cause relatively small decreases of the ongoing ECoG responses (−4.3 dB), such changes may not be detectable in threshold determinations. Additionally, compared with suprathreshold ECoG recordings with larger signals, determinations of detection thresholds are more time consuming and more sensitive to small changes of the electrical background noise, which in our experience often varies during surgery and can conceal or cause changes of the detection threshold.

CONCLUSION

Trauma limited to the high-frequency regions of the cochlea that is not detectable in low-frequency ECoG recordings occurs. Therefore, high-frequency ECoG recordings during cochlear implantation give additional information about cochlear trauma. Threshold changes and changes of the amplitude of the ongoing ECoG near threshold reflect changes of the ongoing ECoG signal at higher intensities and therefore do not increase the detection rate of cochlear trauma.

Overall, four out of six subjects with a hearing loss of >11 dB and all subjects with complete hearing loss showed a decrease of either high- or low-frequency ECoG responses. This suggests that acute cochlear trauma during cochlear implantation is an important predictor for postoperative hearing loss, although in some patients postoperative mechanisms independent of surgical trauma play a role.

Scalar dislocation of the electrode array seems to be associated with a decrease of low-frequency ECoG responses and a complete loss of residual hearing.

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